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=> s friend leukemia or MuFL
L1         5460 FRIEND LEUKEMIA OR MUFL

=> s heterologous or exogenous or foreign or reporter
L2       750333 HETEROLOGOUS OR EXOGENOUS OR FOREIGN OR REPORTER

=> s l2 and l1
L3          222 L2 AND L1

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4           114 DUP REM L3 (108 DUPLICATES REMOVED)

=> s l4 and retrovir?
L5           22 L4 AND RETROVIR?

=> d bib ab 1-22
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approach for the analysis of T cell specificity.

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AN 86063725 EMBASE
DN 1986063725
TI Induction of the early stages of Friend erythroleukemia with helper-free Friend spleen focus-forming virus.
AU Berger S.A.; Sanderson N.; Bernstein A.; Hankins W.D.
CS Ontario Cancer Institute, Department of Medical Biophysics, University of Toronto, Toronto, Ont. M4X 1K9, Canada
SO Proceedings of the National Academy of Sciences of the United States of America, (1985) 82/20 (6913-6917).
CODEN: PNASA6
CY United States
DT Journal
FS 016 Cancer
025 Hematology
047 Virology
LA English
AB The polycythemia-inducing strain of Friend virus (FV-P) causes a multistage erythroleukemia in susceptible mice. FV-P is a complex of two viruses, a replication-competent virus [Friend murine leukemia virus (F-MuLV)] and a replication-defective spleen focus-forming virus (SFFVp). We have addressed directly the role of SFFVp in the induction of the early stages of Friend disease by constructing stocks of SFFVp free of detectable F-MuLV, using a recently described **retroviral** helper-cell line. These preparations are capable of inducing erythroid bursts (vBFU-E) whose inducibility, kinetics, and responsiveness to erythropoietin suggest that they are very similar, if not identical, to the vBFU-E induced by FV-P. Single injections of helper-free SFFVp had no apparent effects in vivo, although the addition of **exogenous** helper virus to the inoculum resulted in the induction of classic Friend disease. Increasing the effective titer by giving mice five daily virus injections resulted in the induction of splenomegaly and a large increase in the number of erythroid colony-forming units that were independent of erythropoietin. When the injections were discontinued, the spleens regressed and all the mice survived. When the injections were continued, all the mice died within 25 days of the first injection. These results demonstrate that SFFVp alone can alter the growth characteristics of erythroid progenitors and is directly responsible for the induction of vBFU-E in vitro and the erythroid hyperplasia in vivo. They also demonstrate that the initial polyclonal stage of Friend disease is reversible and can be reproduced by using preparations of SFFVp free of detectable F-MuLV.